

# TUBERCULOUS LIVER ABSCESS IN A CASE WITHOUT LUNG INVOLVEMENT

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Hepatic tuberculosis is an uncommon form of extrapulmonary tuberculosis, particularly when it presents in the form of liver abscess. Here, we report a 64-year-old man who was admitted to our hospital having experienced intermittent chills for 3 months. Aspiration of the liver abscess revealed neither bacteria nor acid-fast bacilli, but pus and granulation tissue were found. Antituberculous therapy was started empirically and cultures of the abscess confirmed the presence of *Mycobacterium tuberculosis* 3 weeks later. We suggest that tuberculous liver abscess should be considered in patients not showing typical features or who fail to respond to antibiotics.

**Key Words:** abscess, liver, tuberculosis  
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Hepatic tuberculosis is an uncommon form of extrapulmonary tuberculosis. It is usually a disseminated disease associated with miliary tuberculosis, which is one of the most characteristic manifestations of tuberculosis [1]. Localized tuberculosis of the liver in the form of macronodular tuberculoma or an abscess is rare. However, because of the increasing incidence of pulmonary tuberculosis, clinicians should be aware of the possibility of tuberculous infection in all patients who have non-resolving liver abscesses [2], particularly in regions with high prevalence, such as Taiwan. We describe a rare case of tuberculous liver and left subphrenic abscesses with no evidence of lung involvement.

## CASE PRESENTATION

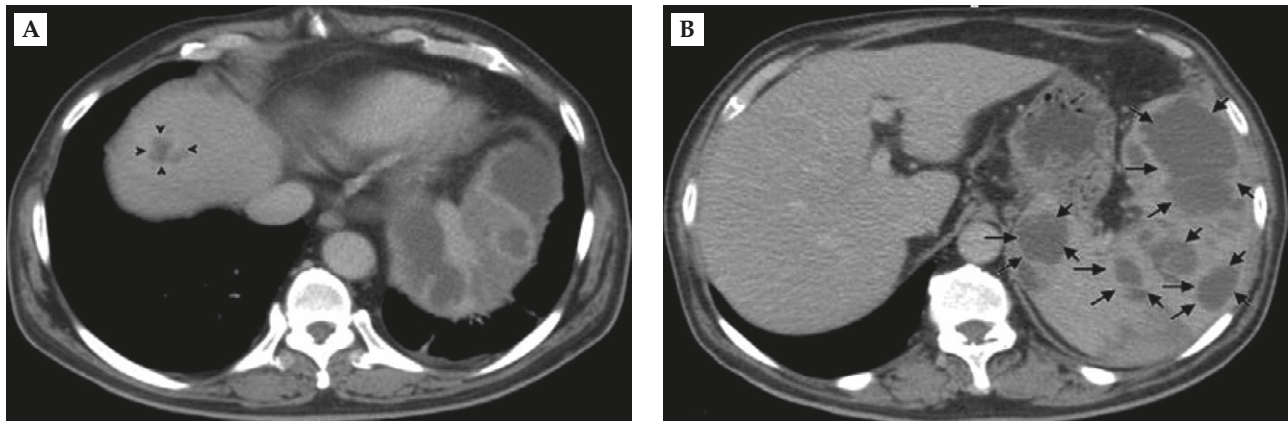
A 64-year-old man with a history of hypertension, with regular treatment, was admitted to our hospital

having experienced intermittent chills for 3 months. He was a retired laborer, and his symptoms included chills, abdominal fullness, and general weakness, which had started 3 months before admission. Body weight loss of 3 kg in the past month was also noted. He had no history of fever, cough, hemoptysis, abdominal pain, or night sweating. Other medical history, travel history, and family history were unremarkable. Because of the above-mentioned symptoms, he was transferred to another hospital where an abdominal computed tomography (CT) scan showed gallstones, liver, and spleen abscesses (Figure 1). Physical examination revealed a soft abdomen without rebounding pain or positive Murphy's sign. Cholecystectomy and splenectomy were done, and pathology showed a multifocal spleen abscess with granulomatous formation. Several sets of acid-fast stains, cultures of *Mycobacterium tuberculosis* and bacteria showed no specific finding. The patient received outpatient treatment followed by oral antibiotics. Unfortunately, his condition worsened with intermittent chills and body weight loss, and he was referred to our hospital.

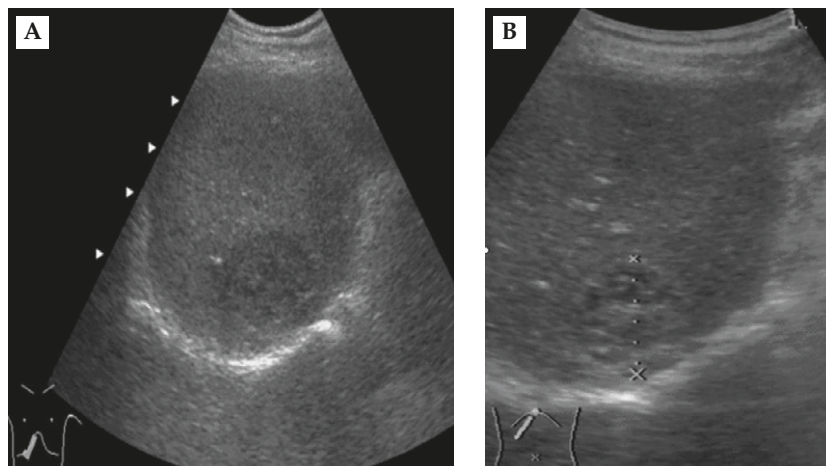
On physical examination, the patient was in mild distress. His body temperature was 36.8°C, heart rate was 91 beats/min, blood pressure was 135/89 mmHg, respiratory rate was 18 breaths/min, and oxygen



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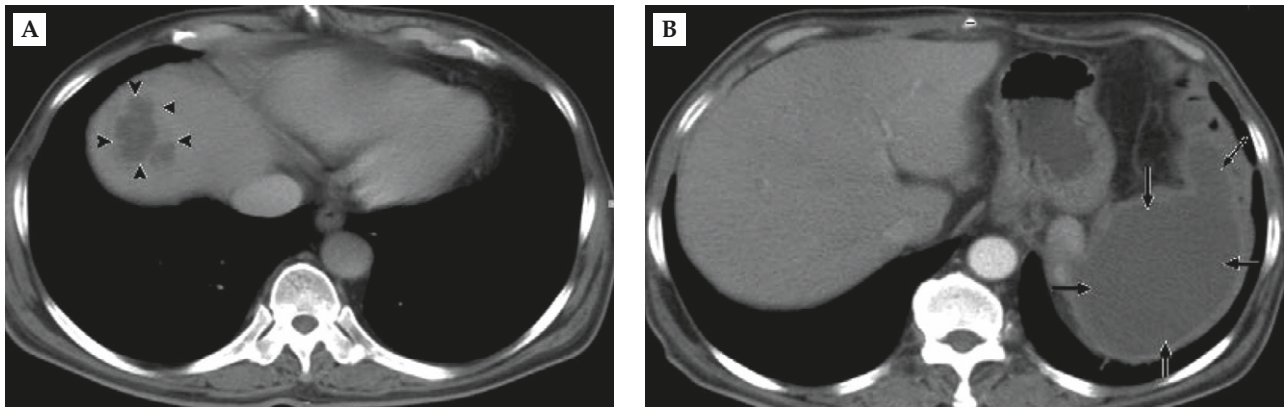
**Figure 1.** Abdominal computed tomography performed 3 months earlier at another hospital showed a liver abscess (arrowhead) and a spleen abscess (arrow).



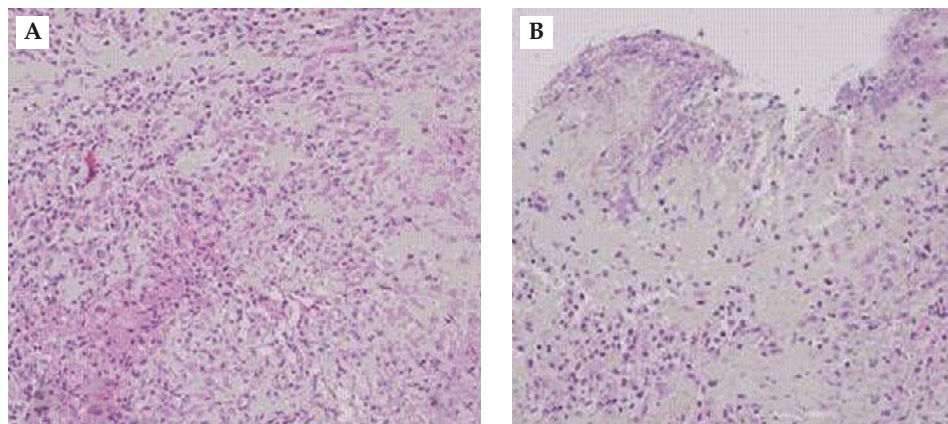
**Figure 2.** Abdominal sonography at admission revealed (A) one hypoechoic mass over the right hepatic lobe, (B) which was resolved 3 weeks after therapy.

saturation was 100% under normal conditions. There was no significant icteric sclera or yellowish skin. No abdominal tenderness or lymphadenopathy was found. The chest X-ray film was normal. An abdominal ultrasound revealed one heterogenous, hypoechoic, irregular mass over the right hepatic lobe, so liver and left subphrenic abscesses were considered (Figure 2A). Abdominal CT revealed a mass lesion with central necrosis in the dome of the liver, and another thick-walled fluid-filled space in the left subphrenic region (Figure 3). Laboratory studies revealed a white blood cell count of  $11.3 \times 10^3/\mu\text{L}$  (normal range,  $4\text{--}11 \times 10^3/\mu\text{L}$ ); hemoglobin of 9.7 g/dL (normal range, 11.3–15.3 g/dL); platelet count of  $589 \times 10^3/\mu\text{L}$  (normal range,  $120\text{--}320 \times 10^3/\mu\text{L}$ ); blood urea nitrogen of

11 mg/dL (normal range, 8–20 mg/dL); creatinine of 0.9 mg/dL (normal range, 0.6–1.2 mg/dL); alanine aminotransferase of 15 U/L (normal range, 8–38 U/L); aspartate aminotransferase of 9 U/L (normal range, 4–44 U/L); alkaline phosphatase of 177 U/L (normal range, 50–190 U/L), and C-reactive protein of 5.6 mg/dL (normal, <0.5 mg/dL). Prothrombin time was within normal limits. Further evaluations, including tests for human immunodeficiency virus, antinuclear antibody, carcinoembryonic antigen, cancer antigen 19–9,  $\alpha$ -fetoprotein, prostate-specific antigen, and amoebic hemagglutination were all negative. Several sets of blood and sputum cultures were negative for bacteria, fungus, and acid-fast bacilli. Percutaneous drainage for the left subphrenic abscess was done, and aspiration



**Figure 3.** Abdominal computed tomography at admission showed an enlarged liver abscess (arrowhead) and a left subphrenic abscess (arrow).



**Figure 4.** Aspiration of the liver abscess revealed (A) granulation tissue with (B) necrosis (hematoxylin and eosin staining; original magnification, 100×).

of the liver abscess revealed pus and granulation tissue (Figure 4). The results of bacterial culture and acid-fast stain in the liver and left subphrenic abscesses were negative. However, based on the clinical appearance, imaging, and pathological findings, abscess of the liver and left subphrenic space due to *M. tuberculosis* was highly suspected, and antituberculous therapy was started empirically. After starting therapy, the patient's condition improved, and laboratory data showed normal white blood cell count and C-reactive protein level at 3 weeks. Three weeks later, cultures of the liver and subphrenic abscesses confirmed the presence of *M. tuberculosis*. However, *M. tuberculosis* was not present in blood and sputum cultures. A repeated sonography of the abdomen 3 weeks post-therapy demonstrated resolution of the liver abscess (Figure 2B). The patient was later discharged from the hospital and received clinical follow-up.

## DISCUSSION

*M. tuberculosis* is the leading cause of death among patients with infectious diseases, but hepatic tuberculosis is a rare form of extrapulmonary tuberculosis [3]. The reason for this may be the low tissue oxygen level, which makes the liver an inhospitable site for this bacillus [4].

In 1929, Rolleston and McNee classified hepatic tuberculosis into two forms, miliary and local [5]. In 1990, Reed et al described three morphological types of hepatic tuberculosis: miliary tuberculosis of the liver, primary miliary tuberculosis of the liver without involvement of other organs, and primary tuberculous abscess of the liver [6]. Levine et al reported a classification similar to that of Reed et al, but added two more types: pulmonary tuberculosis with liver involvement and tuberculous cholangitis [7]. Liver abscess without



pulmonary involvement, such as our case, accounts for only 0.34% of all cases of hepatic tuberculosis [8].

The symptoms and signs of hepatic involvement are usually non-specific, as in our case. An elevated alkaline phosphatase level is the most commonly noted abnormality [9]. Sonographic findings of hepatic tuberculosis usually include hypoechoic lesions [10], but they cannot be differentiated from carcinoma. Contrast-enhanced CT findings include miliary, hypodense nodular or cystic lesions with or without ring enhancement [11]. These radiological findings of tuberculous liver abscess have a low specificity and are of little help in making a definitive diagnosis. Thus, a high level of suspicion is needed for early diagnosis. In patients with liver abscess who do not show typical features and who do not respond to antibiotics or amoebicidal drugs, the possibility of tuberculous abscess should be considered [12].

Radiologically guided diagnostic aspiration with staining for acid-fast bacilli and culture for *M. tuberculosis* help in confirming the diagnosis. However, acid-fast bacilli are only detected in 0–45% of tuberculous liver abscess. The ratio of positive cultures of *M. tuberculosis* in liver abscess is as low as 10%, but is as high as 60% in cases with miliary tuberculosis [7]. Histopathological examination of the specimens from lesions is essential for definitive diagnosis, which depends on the presence of caseating granulomatous lesions or acid-fast bacilli. According to criteria established by Maharaj et al [13], the diagnosis of hepatic tuberculosis should be made by: (1) acid-fast bacilli in liver tissue; (2) tubercle bacilli elsewhere, plus hepatic granuloma with or without Langhans-type giant cells, and/or caseation; (3) typical macroscopic appearance on laparotomy or peritoneoscopy; and (4) response to antituberculous therapy [2]. Thus, the findings of the hepatic histological examination and the good response to antituberculous medications in our patient led us to the early diagnosis of tuberculous liver abscess. Aside from these approaches, polymerase chain reaction assays were also shown to be useful for the early diagnosis of hepatic tuberculosis [14].

In addition to hepatic tuberculosis, some other infectious and non-infectious diseases, such as leprosy, sarcoidosis, Hodgkin's disease, brucellosis, infectious mononucleosis, inflammatory bowel disease, drug-induced liver damage and syphilis, can cause caseating hepatic granulomatous lesions, and these should be considered in the differential diagnosis [15].

Antituberculous therapy is generally recommended for 1 year, and combined percutaneous drainage of the abscess has been advocated [16]. Surgery is reserved for multiple large lesions and lesions refractory to medical treatment.

In conclusion, tuberculosis should be considered in the differential diagnosis of liver abscess, particularly in endemic regions with a high prevalence of *M. tuberculosis* infection, and in patients who do not respond to antibiotics or amoebicidal drugs. The prognosis of liver abscess is good if it is diagnosed early and effective treatment is administered.

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# 無肺部感染徵狀的結核性肝膿瘍

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結核菌造成的肝臟感染病例不常見，尤其是以肝膿瘍型態表現者。我們報告一位 64 歲男性病例因為 3 個月間斷性發冷的症狀而來本院住院。檢查發現有肝膿瘍，經細針抽吸未培養出細菌或抗酸菌，病理檢查發現有肉芽組織。針對這位病例預防性投與抗結核菌藥物，而 3 星期後膿瘍培養證實為結核桿菌感染。我們的結論是肝膿瘍患者如果有不典型表現或對抗生素治療無效時，結核性肝膿瘍應該要列入考慮。

**關鍵詞：**肝膿瘍，結核桿菌  
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